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Original Article

Analysis of Vestibular Evoked Myogenic Potentials and Electrocochleography in Noise Induced Hearing Loss

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OBJECTIVE: Our objective was to analyze the electrocochleography (ECoG) and cervical vestibular evoked myogenic potential (cVEMP) results of patients with noise-induced hearing loss (NIHL).

MATERIALS and METHODS: The study included 20 patients with NIHL. Pure-tone audiometry, tympanic membrane ECoG, and cVEMP were performed on all patients. The patients were divided into two groups based on averaged thresholds at 4, 6, and 8 kHz; whereby, group 1 comprised patients who had a threshold higher than 68.3 dB HL, whereas group 2 comprised patients with a threshold lower than 68.3 dB HL.

RESULTS: Group 2 had a significantly higher number of patients with abnormal cVEMP values (63% versus 28%) ($p=0.028$). There was no significant difference in the incidence of ECoG abnormality between the groups ($p>0.05$), but there was a significant difference in the incidence of recognizable ECoG potentials between the groups ($p<0.05$). When only patients with vertigo/dizziness were considered, the group with vertigo and a lower degree of hearing loss (group 2) showed a higher incidence of abnormal cVEMP ($p<0.05$).

CONCLUSION: Although the anatomical proximity of the sacculus to the cochlea leads to the consideration of a common involvement of these structures in NIHL, our results did not support the idea of a common and proportional involvement of the vestibular and auditory systems. Our study shows that saccular involvement is disproportionate to auditory involvement in NIHL.

KEYWORDS: Noise-induced hearing loss, vestibular evoked myogenic potential, electrocochleography

INTRODUCTION

Noise-induced hearing loss (NIHL) can occur due to acute or chronic acoustic overexposure. Environmental and occupational conditions are two of the most common contributing factors to the development of chronic NIHL. Cochlear damage caused by acoustic trauma usually results in hearing loss and tinnitus. Similar to the effects of acoustic trauma on the cochlea, some parts of the vestibular system, such as the sacculus, may also be affected [1, 2]. In this circumstance, vestibular symptoms can appear but may be neglected [3, 4]. To investigate the effects of chronic acoustic trauma, electrocochleography (ECoG) and cervical vestibular evoked myogenic potential (cVEMP) can be used, besides performing basic audiological tests. While ECoG evaluates the cochlear hair cells and distal part of the auditory nerve by means of summation and action potentials, cVEMP is used for the evaluation of the vestibular system, especially the sacculus.

In this study, we analyzed the ECoG and VEMP results as well as the signs and symptoms in patients with chronic NIHL.

MATERIALS and METHODS

The study was conducted in Gülhane Military Medical Academy, Department of Otolaryngology, Head and Neck Surgery. The study protocol was approved by Gülhane Military Medical Academy ethics committee (Ethics committee no: 06/10/2010-160). The study comprised 20 patients with NIHL. The diagnosis of NIHL was based on the criteria of the American College of Occupational and Environmental Medicine [5]. Each patient had a long-term history of acoustic trauma. All study participants provided informed written consent prior to study enrollment. All patients were males; they were military personnel. The detailed history collected from each patient included questions on how many years they had been exposed to noise and whether they had vertigo, dizziness, or tinnitus. Pure-tone audiometry, tympanic membrane ECoG, and cVEMP were performed on all patients. Pure-tone audiometry included air conduction thresholds at the frequencies of 0.25, 0.5, 1, 2, 4, and 8 kHz, and bone-conduction thresholds at 0.5, 1, 2, and 4 kHz.

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Only patients with bilaterally symmetrical hearing loss at 3, 4, or 6 kHz specific to noise exposure were included. Patients with any other otological disease, such as tympanic membrane perforation, middle or outer ear canal pathology, or asymmetrical hearing loss, were excluded from the study. The ages of the patients ranged from 18 years to 66 years. The mean age was 41.2±9.24 years.

Audiometry

The pure-tone thresholds in patients with NIHL characteristically showed a notch centered at 3, 4, or 6 kHz. For quantification, the NIHL hearing thresholds at 4, 6, and 8 kHz were calculated because the averaged thresholds at these frequencies better reflect the degree of hearing loss [6]. The mean pure-tone average at 0.5, 1, and 2 kHz was 15.2±5.65 dB, 15.7±6.45 dB, and 27.6±19.90 dB, respectively. The averaged threshold at 4, 6, and 8 kHz was 68.3 dB HL. The patients were grouped based on this averaged threshold: group 1, where the patients had a threshold higher than 68.3 dB HL, and group 2, where they had a threshold lower than 68.3 dB HL.

ECoG

ECoG recording was performed in a quiet room specifically designed for auditory evoked potentials using a Smart-EP Multi-Channel Evoked Potential system (Windows Version 2.0, IHS Co. FL, USA) and tiptrode tympanic membrane electrode (Biologic Co., IL, USA). The patients were placed in the supine position. Monopolar disk electrodes were used as reference and ground electrodes. The reference electrode was ipsilaterally placed on the mastoid bone, and the ground electrode was placed on the upper forehead. Sound stimuli were delivered through a earphone (ER-3A™, Etymotic Research Inc., Elk Grove Village, Illinois, USA) inserted into the ear canal. The fixation of the recording electrode was achieved by placing the foam tip of the earphone into the ear canal.

Click stimuli of 90 dB nHL in alternating polarity were applied. The number of click stimuli ranged from 250 to 700 based on the wave formation quality. The stimulus repetition rate was 9.7/s. Low- and high-pass filters were set at 10 Hz to 3 kHz. Potentials were amplified with a gain of 10^5. The summation potential (SP) and action potential (AP) were recognized on traces, and SP/AP was calculated following the identification of the peaks of the potentials. The upper cut-off value of SP/AP was calculated by adding +2 standard deviation to the mean SP/AP (0.22+2×0.05=0.32) obtained from the control subjects of the laboratory [7]. SP/AP and waveform morphology were checked on the obtained double traces. The traces were labeled as recognizable or not recognizable based on the waveform morphology. Those values exceeding the cut-off value of 0.32 and those traces with no identifiable waves (labeled as recognizable wave absent) were considered abnormal.

cVEMP

Medelec Synergy version 10 (VIASYS HealthCare UK, Surrey, UK) and TDH-39 headphones (Telephonics Co., NY, USA) were used for the testing. The surface electromyographic activity was recorded from the upper half of each sternocleidomastoid muscle (SCM) with a reference disk electrode on the upper sternum. During the recording, the patient was in a sitting position and was instructed to rotate his/her head to the opposite side of the stimulated ear. The signal was 110 dB nHL click stimulus, and the responses to 200 stimuli were averaged twice for reliability control. The peak-to-peak amplitude of the first positive-negative waves (P13–N23) was measured. The averaged latency in two runs was regarded as the latency of P13 and N23. Using normative data obtained by one of the authors (OY) in the laboratory, the mean±2 standard deviation (SD) was considered the upper cut-off of the normal range [8]. Those values exceeding the mean±2SD (P13:13.74 ms±2SD=14.65, N23: 21.91 ms±2SD=23.56) or traces with no clear wave formation were classified as abnormal VEMP responses.

Statistical Analysis

The SPSS 15.0 for Windows program was used for statistical analysis (IBM SPSS Statistics, IBM Corporation; Chicago, IL, USA). Pearson's chi-square and Fisher's exact tests were used to compare categorical data, and Mann–Whitney U test was used to compare continuous data between the groups. A value of $p<0.05$ was accepted as statistically significant.

RESULTS

Twenty male patients (40 ears) were included in the study. Group 1 consisted of 21 ears and group 2 consisted of 19 ears. The mean ages of the patients in groups 1 and 2 were 37.86±5 and 44.56±11.8 years, respectively. The mean duration of noise exposure in groups 1 and 2 were 16.67±4.46 and 15.5±5.19 years, respectively. There was no significant difference in age and the duration of noise exposure between the groups ($p>0.05$, Mann–Whitney U test) (Table 1). There was no significant difference in the incidence of tinnitus, vertigo, or dizziness symptoms between the groups ($p>0.05$, chi-square and Fisher's exact tests) (Table 1).

The SP, AP, SP/AP, P13, and N23 values for both groups are presented in Table 2. There was no significant difference in the SP, AP, and SP/AP between the groups ($p>0.05$, Mann–Whitney U test). However, group 1 had significantly shorter VEMP latencies ($p<0.05$, Mann–Whitney U test) (Table 2).

Overall, an abnormal ECoG was recorded from 35 patients (87.5%), irrespective of the grouping. There was no significant difference in the incidence of ECoG abnormality between the groups ($p>0.05$, Pearson's chi-square and Fisher's exact tests) (Table 3).

Overall, of all patients, 18 (45%) showed abnormal cVEMP, irrespective of the grouping (Figure 1). As for the group-specific cVEMP re-

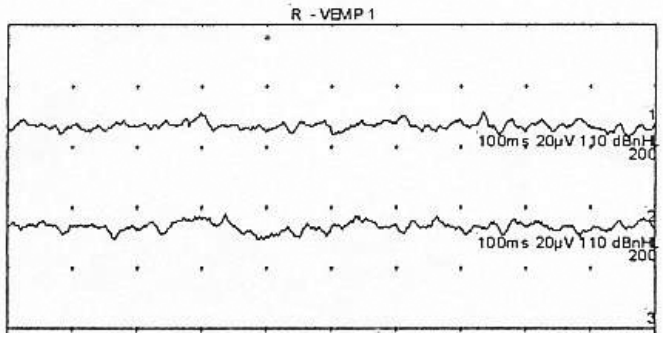

Figure 1. No identifiable VEMP in group 1

Table 1. Comparison of groups with reference to age, threshold of hearing, duration of noise, incidence of tinnitus, dizziness, and vertigo

		Group 1			Group 2				
		Mean±SD	Min.	Max.	Mean±SD	Min.	Max.		p value*
Age		37.8±5	28	44	44.5±11.81	31	66		0.137
Averaged threshold at 4, 6, and 8 kHz		83.9±6.78	73.3	96.6	51.0±13.59	20	66.6		0.000
Duration of noise exposure (year)		16.6±4.46	9	23	15.5±5.19	10	25		0.479
			n	(%)	n	(%)	n (Total)	(%)	
Tinnitus	Present		17	(81.0)	17	(89.5)	34	(85.0)	0.664
	Absent		4	(19.0)	2	(10.5)	6	(15.0)	
Dizziness	Present		9	(42.9)	5	(26.3)	14	(35.0)	0.273
	Absent		12	(57.1)	14	(73.7)	26	(65.0)	
Vertigo	Present		11	(52.4)	7	(36.8)	18	(45.0)	0.324
	Absent		10	(47.6)	12	(63.2)	22	(55.0)	

*Mann–Whitney U test

Bold p value is statistically significant.

Table 2. Comparison of groups with reference to the electrocochleography and vestibular evoked myogenic potential tests

	Group 1			Group 2			
	Mean±SD	Min.	Max.	Mean±SD	Min.	Max.	p value*
SP	0.1±0.06	0.02	0.2	0.13±0.12	0.01	0.47	0.751
AP	0.24±0.14	0.11	0.46	0.23±0.18	0.07	0.67	0.812
SP/AP	0.46±0.19	0.18	0.67	0.55±0.24	0.17	0.95	0.362
P13	13.63±1.13	12	16.6	14.47±1.25	11.9	16.2	**0.034**
N23	21.68±1.19	20.2	24.3	23.15±1.73	19.8	25.5	**0.024**

*Mann–Whitney U test

Bold p values are statistically significant.

SP: Summation potential; AP: action potential; P13: positive waves; N23: negative waves

Table 3. Vestibular evoked myogenic potential and electrocochleography in groups

		Groups			p
		Group 1 n (%)	Group 2 n (%)	Total n (%)	value *
ECoG	Abnormal response	19 (90.5)	16 (84.2)	35 (87.5)	0.654
	Normal response	2 (9.5)	3 (15.8)	5 (12.5)	
	Total	21 (52.5)	19 (45.7)	40 (100.0)	
VEMP	Abnormal response	6 (28.6)	12 (63.2)	18 (45.0)	0.028
	Normal response	15 (71.4)	7 (36.8)	22 (55.0)	
	Total	21 (52.5)	12 (47.5)	40 (100.0)	

*: Pearson's chi-square and Fisher's exact tests

VEMP: vestibular evoked myogenic potential; ECoG: electrocochleography

sults, there was a significant difference in the incidence of abnormal cVEMP between the groups (p=0.028, Pearson's chi-square and Fisher's exact tests). Group 2 had significantly more patients with abnormal cVEMP (63% versus 28% in group 1) (Table 3).

Recognizable ECoG potentials were obtained in 7 patients in group 1 (Table 4), of whom 5 had abnormal SP/AP values (Figures 2 and 3). Group 2 had 13 patients with recognizable ECoG potentials, and of these, only 3 had normal SP/AP values (Figure 4). There was a significant difference in the incidence of recognizable ECoG potentials between the groups (p<0.05, Pearson's chi-square and Fisher's exact tests) (Table 4). There was no significant difference in the incidence of recognizable cVEMP between the groups (p>0.05, Pearson's chi-square and Fisher's exact tests) (Table 4 and Figure 5).

A final comparison was made in patients with only vertigo/dizziness. In this comparison, the group who had vertigo and a lower degree of hearing loss (group 2) showed a higher incidence of abnormal cVEMP results (p<0.05, Pearson's chi-square and Fisher's exact tests) (Table 5).

DISCUSSION

Some cellular changes occur in the inner ear(s) of patients with NIHL. These changes may be due to direct mechanical trauma or metabolic changes. The causes of damage are ischemia, reactive oxygen radicals, and metabolic overload in the organ of Corti [9, 10]. A prolonged

Table 4. Comparison of groups with reference to recognizable vestibular evoked myogenic potential and electrocochleography

		Groups			p
		Group 1 n (%)	Group 2 n (%)	Total n (%)	value *
Recognizable ECoG potentials	Present	7 (33.3)	13 (68.4)	20 (50.0)	**0.027**
	Absent	14 (66.7)	6 (31.6)	20 (50.0)	
Recognizable VEMP	Present	16 (76.2)	12 (63.2)	28 (70.0)	0.369
	Absent	5 (23.8)	7 (36.8)	12 (30.0)	
	Total	**21 (52.5)**	**19 (47.5)**	**40 (100.0)**	

*: Pearson's chi-square and Fisher's exact tests
Bold p value is statistically significant
VEMP: vestibular evoked myogenic potential; ECoG: electrocochleography

Table 5. Comparison of groups with reference to vestibular evoked myogenic potential in patients who had vertigo/dizziness

		Groups			p
		Group 1 n (%)	Group 2 n (%)	Total n (%)	value *
VEMP	Abnormal Response	2 (18.2)	5 (71.4)	7 (38.9)	**0.049**
	Normal Response	9 (81.8)	2 (28.6)	11 (61.1)	
	Total	**11 (61.1)**	**7 (38.9)**	**18 (100.0)**	

*: Pearson's chi-square and Fisher's exact tests
Bold p-value is statistically significant
VEMP: vestibular evoked myogenic potential

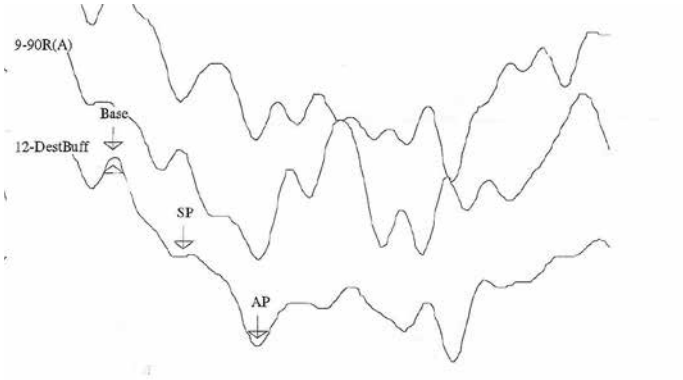

Figure 2. ECoG traces in group 1. SP/AP is 0.53. (SP: summation potential, AP: action potential)

duration of noise exposure may cause permanent damage [11]. A characteristic audiological finding of NIHL is high-frequency hearing loss, especially at 4 kHz. In the early stages of NIHL, almost 30 dB hearing loss at 4 kHz frequency can be seen, and this loss is caused by damage in the related place in the organ of Corti [11]. Histopathological studies have shown that long-term noise exposure causes cochlear damage, specifically at a 9 to 13 mm-area of the cochlea. This site is responsible for the 4 kHz frequency response [12]. Therefore, we assessed the frequencies of 4 to 8 kHz in the present study.

Vestibular structures can be affected by noise due to their anatomical proximity. Early studies showed that the sacculus and the cochlea

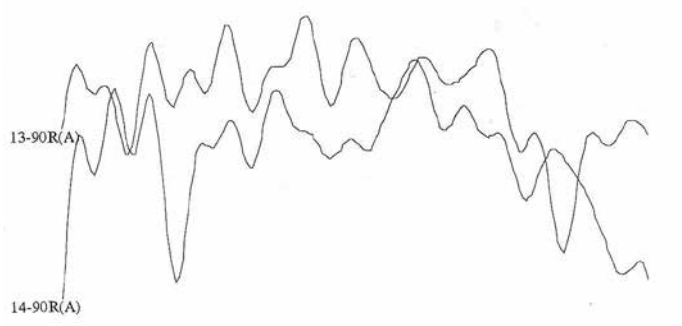

Figure 3. No identifiable ECoG potential in group 2

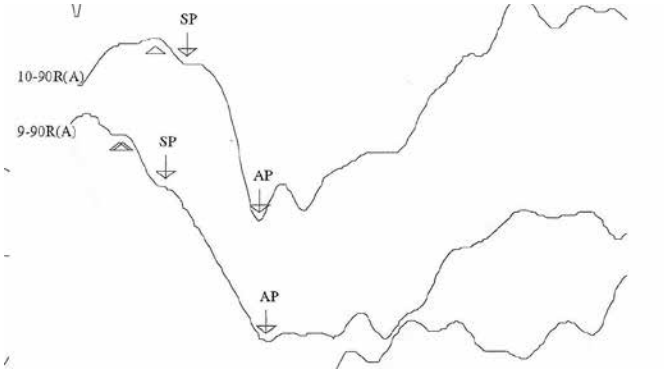

Figure 4. ECoG traces in group 2. SP/AP is 0.26

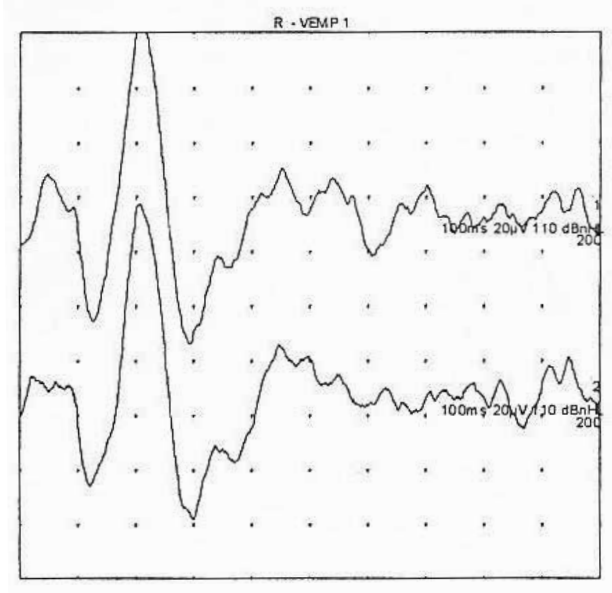

Figure 5. Normal VEMP traces in group 2

(pars inferior) can be affected by noise, whereas the utriculus and semicircular canal are not [2, 3, 13, 14]. There is no postmortem study on the sacculus's involvement in NIHL. Even though there are studies showing certain kinds of changes in the sacculus in human cadavers with sensorineural hearing loss (SNHL), the relationship between SNHL and the sacculus has not been established [15].

The vestibular system, especially sacculus damage caused by noise, emerges by the same mechanism [9, 10, 13]. The cVEMP response covers

the sacculus, inferior vestibular nerve, vestibular nucleus, and cervical muscle activity [3]. Wang et al. [3] and Sazgar et al. [16] showed that there is a relation between vertigo and the VEMP results in acoustic trauma. Wang et al. [3] found abnormal VEMP in 50% of twenty patients with NIHL. They stated that hearing loss at >40 dB at 4 Hz is associated with VEMP abnormality. However, in our study, group 2 had better hearing, yet had significantly prolonged VEMP and also a higher incidence of VEMP abnormality.

The analysis of VEMP in patients with vertigo/dizziness yielded contradictory results. The assessment of VEMP in patients with vertigo/dizziness only showed that patients who had less hearing loss (group 1) had a higher incidence of VEMP abnormality. Overexposure to noise can cause a reduction in blood flow in the stria vascularis of the cochlea [17-20] and a reduction in the size of the endocochlear potential [21, 22]. There is no consensus on as to what degree of vestibular structures is affected by a given noise. Some studies on patients with NIHL show abnormal results in VEMP testing [3]. For instance, Kumar et al. [23] found that VEMP was absent or abnormal in 67% of their study patients with NIHL. Hsu et al. [24] showed temporary and permanent VEMP abnormalities along with auditory brainstem response abnormality in an experimental setting. In contrast, the opposite view proposes that there is no proportional relationship between the level of hearing loss and VEMP. Also, vestibular structures would be less affected by noise than the cochlea [17].

Our results showed a higher incidence of VEMP abnormality in the group with a better hearing level. Moreover, P13 and N23 latencies were significantly prolonged in this group than in group 1. On the other hand, even though it was not a significant finding, the higher hearing loss group consisted of more patients with vertigo/dizziness. When we considered patients with vertigo/dizziness in comparison between groups 1 and 2, the better hearing group (group 2) had a higher incidence of VEMP abnormality. We used click stimuli of 90 dB HL for all subjects. Although it is well known that hearing loss could affect ECoG responses, it has been accepted that SP/AP is not affected by the degree of hearing loss. The idea behind ECoG is to show SP and AP in a robust response obtained with a high level of acoustic signals. Defining potentials could have been more difficult if lower levels of acoustic signals had been used. SP originates from inner hair cells, and AP originates from the distal part of the cochlear nerve [25].

It is difficult to determine the effect of noise on the inner ear in NIHL. In brief, ECoG assesses hair cell function relative to cochlear nerve function with the parameter of SP/AP. Even though ECoG is mostly used to detect endolymphatic hydrops, some ECoG studies have shown cochlear cell damage resulting from SNHL and NIHL [26-29]. Thus, we think that ECoG may be helpful to give some information about the effect of NIHL on the inner ear. Nowadays, ECoG recorded via an extra-tympanic electrode has proven to be useful. It is also non-invasive, easy to apply, and painless. Furthermore, it was stated that there was no difference between extra-tympanic and transtympanic ECoG data [30, 31].

In the present study, we found that the degree of hearing loss did not cause any difference in the latencies of SP and AP, SP/AP, or the incidence of abnormal ECoG. However, there was a significant difference in the incidence of recognizable ECoG potentials between groups 1 and 2. As expected, the higher hearing loss group showed a lower number of recognizable ECoG potentials, possibly due to the fact that a higher level of hearing loss might impede the generation of recognizable ECoG. Nam and Won (2004) showed a transient hearing threshold shift and SP/AP changes due to acoustic trauma [29]. Apart from NIHL, the presence of SNHL itself may cause a problem in the generation of robust AP [27, 32]. It seems reasonable to propose that a higher hearing loss may cause more damage to the inner ear and result in abnormal ECoG. Moreover, there may be no ECoG response in the case of higher hearing loss.

To the best of our knowledge, we failed to find another comparative study investigating cochlear and saccular functions by means of ECoG and cVEMP in the case of long-term noise exposure. Although the anatomical proximity of the sacculus to the cochlea leads to the consideration of a common involvement of these structures in NIHL, a common and proportional involvement of the auditory and vestibular systems may not be the case because the recovery of these structures from noise-related changes may not occur to the same degree. Therefore, this factor should be kept in mind while interpreting our results. To conclude, our study shows that saccular involvement is disproportionate to auditory involvement in the case of long-term noise exposure.

Ethics Committee Approval: Ethics committee approval was received for this study.

Informed Consent: Written informed consent has been obtained from all participants.

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